

Hypoplasia of the aortic root¹

The problem of aortic valve replacement

ROWAN NICKS, T. CARTMILL, and L. BERNSTEIN

*Department of Cardio-thoracic Surgery and the Hallstrom Institute of Cardiology,
the Royal Prince Alfred Hospital, Sydney*

We report a technique for the enlargement of a hypoplastic aortic root by an operation whereby the hypoplastic aortic root has been so enlarged by the insertion of a Dacron fabric gusset that it will accommodate a size 9A or larger Starr-Edwards prosthesis. Our experience in five patients is described. No matter what type of valve is used for replacement of a diseased aortic valve, and no matter what improved designs of valvular prosthesis are ultimately developed, it will be necessary (in the particular group described) to enlarge the aortic ring to accommodate a size which will function correctly without causing left ventricular outflow obstruction.

Aortic valve disease frequently is associated with an aortic annulus of smaller than normal size. This is especially likely when calcific aortic stenosis complicates pre-existing congenital aortic stenosis.

Whether the aortic valve is replaced by a prosthesis, an allograft, or a xenograft, a restricted area at the aortic annulus limits the relief of obstruction of the left ventricular outflow.

We believe our experience may be of help to other surgeons confronted with this difficult technical problem.

MATERIAL AND METHODS

We have defined a hypoplastic aortic root as one with an aortic ring and proximal aorta of substantially less than 2.5 cm. in diameter. Such an aortic annulus, after removal of the diseased cusps, will accept a small size (8A) Starr-Edwards prosthesis with difficulty or not at all.

We have carried out an operation designed to enlarge the aortic ring and proximal aorta in such a

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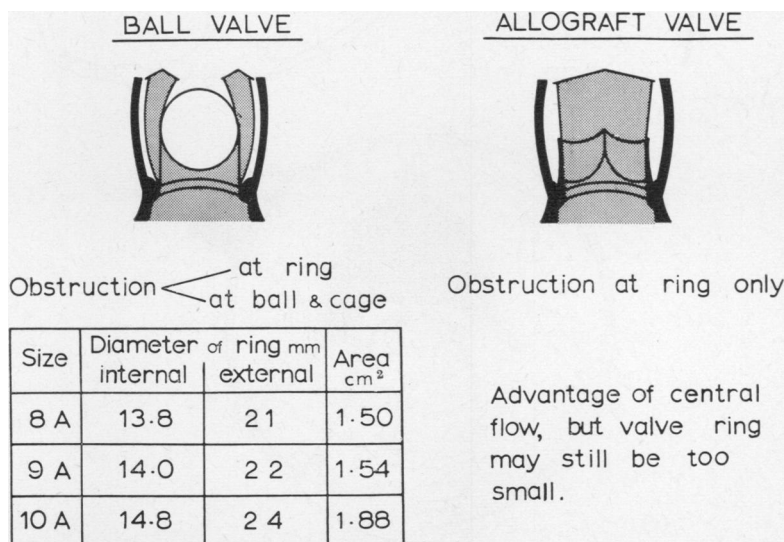


FIG. 1.

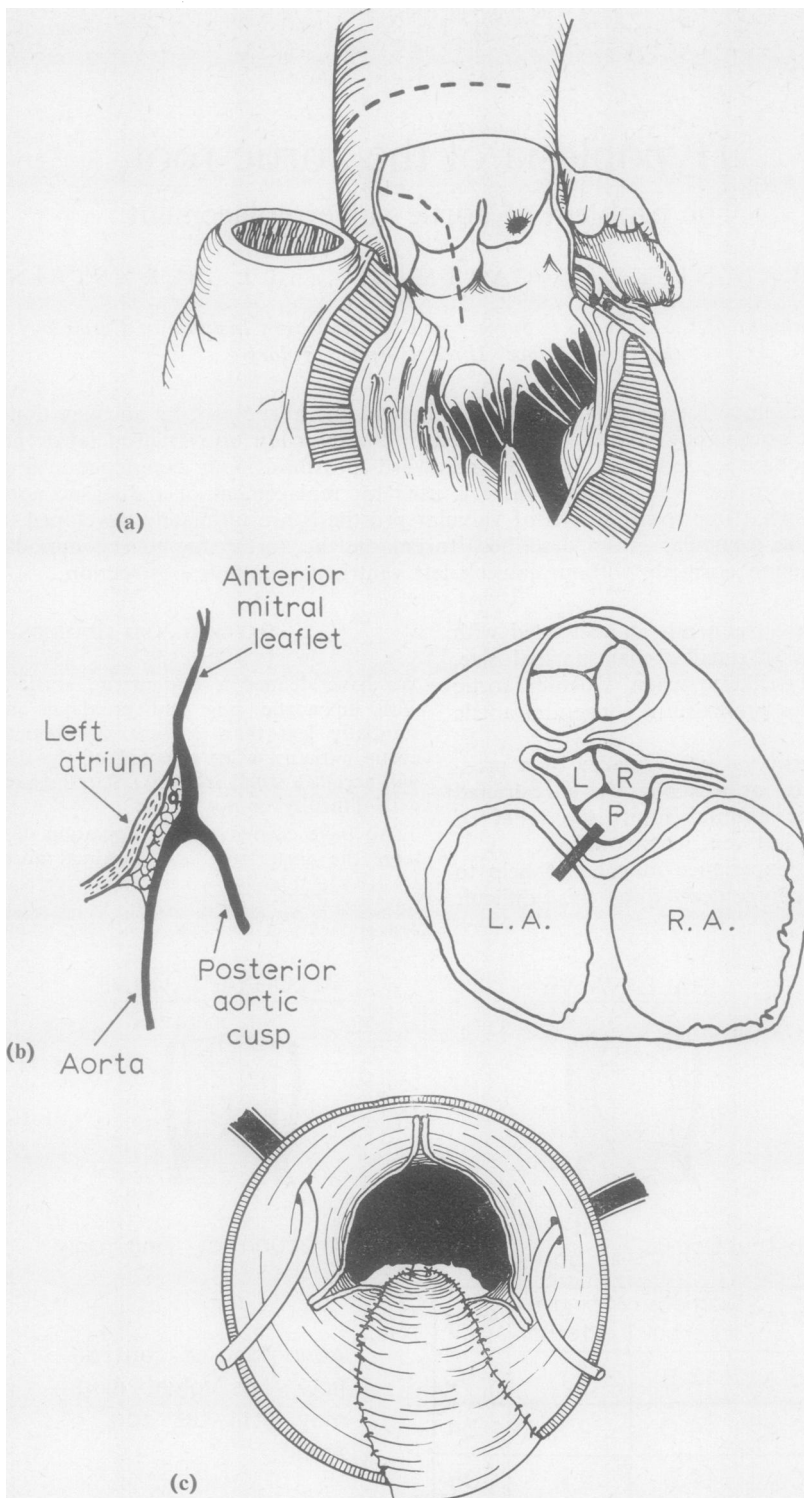


FIG. 2. *For legend see facing page.*

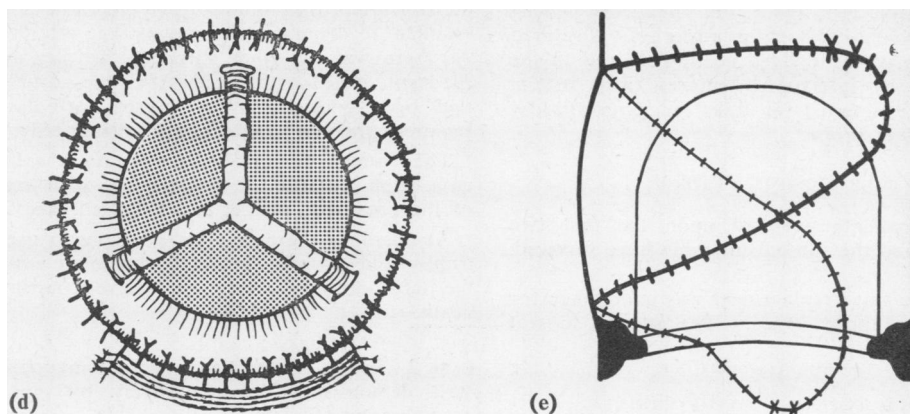


FIG. 2. (a) The incision. (b) Anatomical section of aortic root through posterior sinus of Valsalva and origins of aortic and mitral valves (from above and as the surgeon approaches it). (c) The patch in position. (d) The valve firmly tied down. The arc attached to the patch is posterior. (e) The lie of the patch is shown by the suture lines behind and in front. The valve attachment to the aortic ring and the cage within the aorta are sketched in.

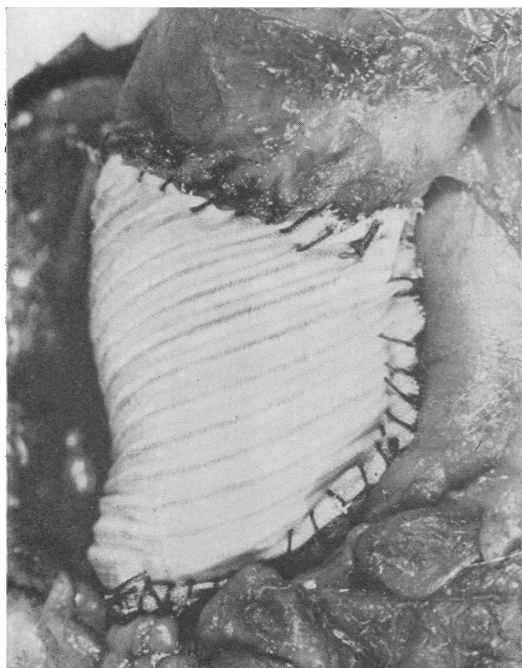


FIG. 3. The Dacron patch in place after insertion of the valve and closure of the aorta.

manner that a size 9 or 10 Starr-Edwards prosthesis may be comfortably fitted without obstruction to the cage or to the ball movement.

This size was chosen as the smallest compatible with a free flow into the aorta and with an easy accommodation of the cage.

We have confined our observations to the Starr-Edwards prosthesis, which we routinely use for aortic valve replacement. The external diameter of the 9A valve is 22 mm., with an orifice area of 1.54 sq. cm., while that of the 10A is 24 mm., with a valve area of 1.88 sq. cm. (Fig. 1). We have preferred to use this latter size, provided it fits satisfactorily.

OPERATION TECHNIQUE (Fig. 2a-e) With the patient on total cardiac bypass, the proximal aorta clamped and the heart beat smoothly sustained by individual coronary artery perfusion at 32° C. (ensured by monitoring flow, line pressure, and the electrocardiogram) the calcified aortic valve cusps are thoroughly excised and the ring-orifice is measured. When it is revealed that the aortic annulus is small, the aortic incision is carried downwards posteriorly through the non-coronary aortic sinus across the aortic ring as far as the origin of the mitral valve (Figs 2a, b and 3).

A tongue of woven Dacron material, cut from a prosthetic tubular arterial graft and with its natural curvature facing the lumen, is sutured down to the fibrous origin of the mitral ring with two mattress sutures. These sutures are then continued along anterior and posterior margins of the aortic incision.

The prosthetic inlay provides a substantial enlargement, both of the aortic ring and of the proximal aorta, so that it can now accept a minimum size 9 or, better, size 10 Starr-Edwards prosthesis.

The valve is then sutured to the aortic ring and tied firmly into position in the usual manner.

When the valve is in place, the unattached sewing ring adjacent to the inlay patch posteriorly is sutured to the margins of the divided aortic ring and to the Dacron inlay by interrupted sutures from within. The suture line is tested for leaks on completion by means of a silver probe.

CASE REPORTS (Table)

Of the five patients operated upon the first two patients died and the subsequent three have survived.

CASE 1 Aortic valve replacement was undertaken for relief of severe aortic stenosis. The attempt to seat

the size 8A prosthesis into the hypoplastic aortic ring resulted in bending the metal cage of the valve. As there was no other valve of this size available, the struts were straightened. After the aortic annulus had been enlarged by a 'gusset aortoplasty', the repaired prosthesis was seated satisfactorily.

The patient was unable to sustain a satisfactory circulation because of interference with movement of the prosthetic poppet and damaged cage.

At necropsy there was marked subvalvular hypertrophy and fibrosis (Fig. 4). A calcium embolus in the left coronary artery may have contributed to death.

CASE 2 In this patient the aortic root hypoplasia was well demonstrated pre-operatively by ciné angiocardiology (Fig. 5).

In 1965 aortic valvotomy with débridement of the calcified valve was performed, and a Dacron patch was inserted to enlarge a supravalvular narrowing of the ascending aorta. He was well for three years until a month prior to this second readmission, at which time he had developed severe heart failure with pulmonary oedema.

After a period of rest and medical treatment re-operation was undertaken. The aortic ring was enlarged by a more extensive inlay and a size 8A prosthesis was inserted. He died 24 hours after operation with a progressive fall in cardiac output.

TABLE
AORTIC HYPOPLASIA—CATHETER DATA

Case	Date	PA	LV	BA	LV-BA	CO	AVA
1 AM 42F 016141	—						
2 DS 28M 200224	11/64 3/67		226 183	131 121	95 62	5.4 3.5	0.8 0.5
3 MM 22F 088080	9/68 3/69	21 13	108 130	108 100	0 30	2.6 4.5	
4 VK 46F 260433	11/68	38	167	145	22	3.1	
5 EG 41F 172129	4/66 4/69	18	187 151	126 134	61 17	4.2 4.2	0.7 1.6

PA=pulmonary artery pressure (mm. Hg); LV=systolic pressure in left ventricle (mm. Hg); BA=systolic pressure in brachial artery (mm. Hg); CO=cardiac output (l./min.); AVA=calculated aortic valve area (cm.²).



FIG. 4. Case 1. Necropsy specimen showing an 8A Starr-Edwards prosthesis firmly fixed in position with the mitral origin below. There is marked subvalvular hypertrophy and fibrosis of the septum.

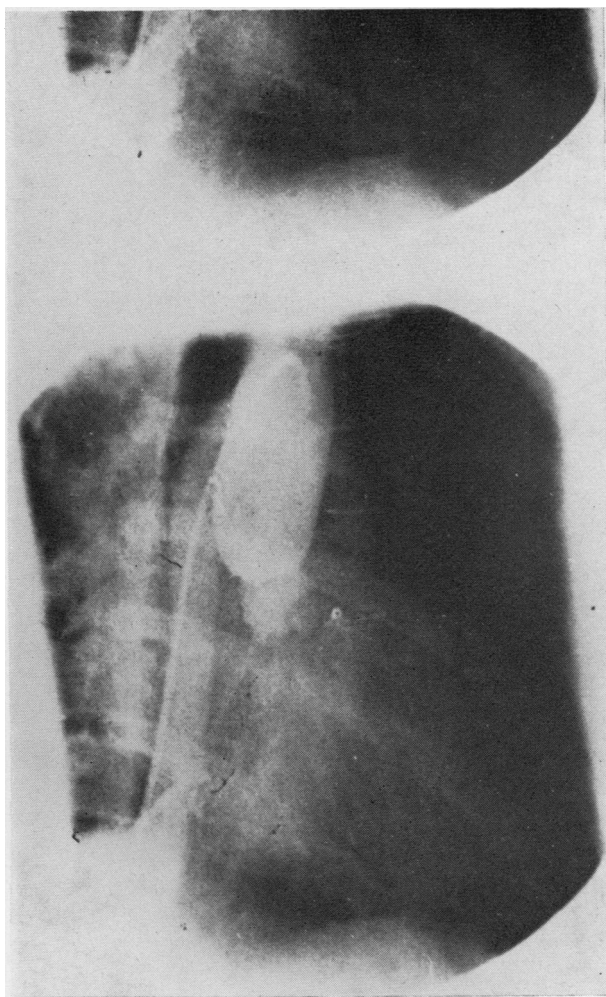


FIG. 5. Case 2. Ciné angiogram showing hypoplasia of the aortic root.

The specimen, which had been badly fixed in the coroner's department, showed the aortic valve sutured to the ring and the gusset. The prosthesis was well seated, and there was adequate clearance allowing free movement of the poppet. There was marked subvalvar muscular hypertrophy partly obstructing the prosthetic orifice. The histological sections revealed extensive replacement fibrosis of the myocardium.

After these experiences, and further study of the previous necropsy specimens, it was decided that on the next occasion we would proceed immediately with the insertion of a large gusset so as to permit the fitting of a size 9A or preferably 10A prosthesis.

The next three patients survived the operation.

CASE 3 In this patient, aged 22 years, a congenitally stenosed and incompetent aortic valve was replaced by a freeze-dried, irradiated aortic valve homograft.

The valve functioned satisfactorily for six months. Cusp disintegration occurred quite suddenly (Fig. 6), causing severe aortic incompetence and heart failure.

At a second operation, a size 9A Starr-Edwards prosthesis was inserted after enlarging the aortic root in the manner described. The patient made a good recovery. Cardiac catheterization performed nine months post-operatively showed mild aortic stenosis with a gradient of 30 mm. across the valve, while ciné aortography demonstrated a mild aortic incompetence through a periannular leak.

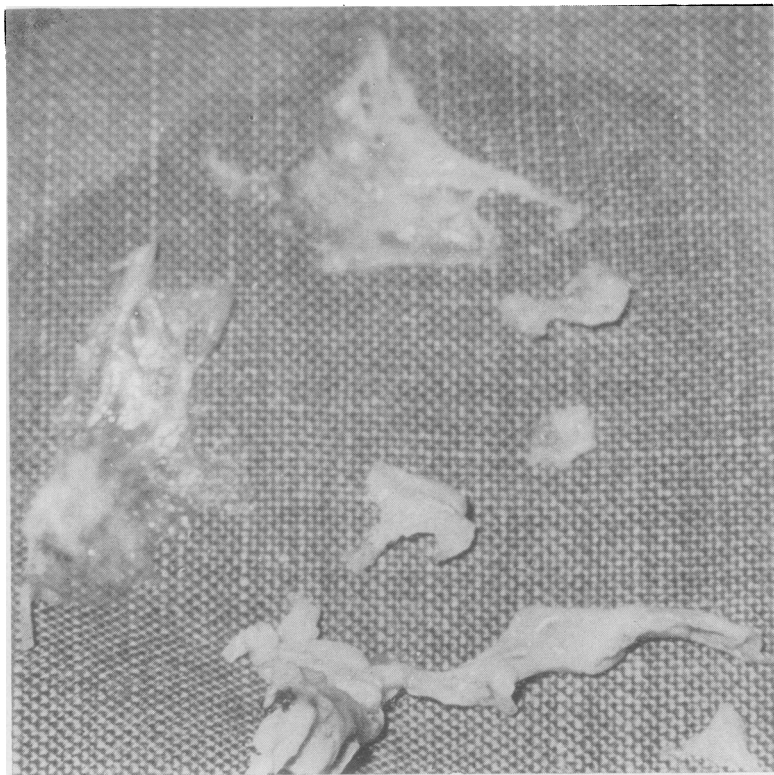


FIG. 6. *Case 3. The disintegrated homograft cusps removed at a second operation five months later.*

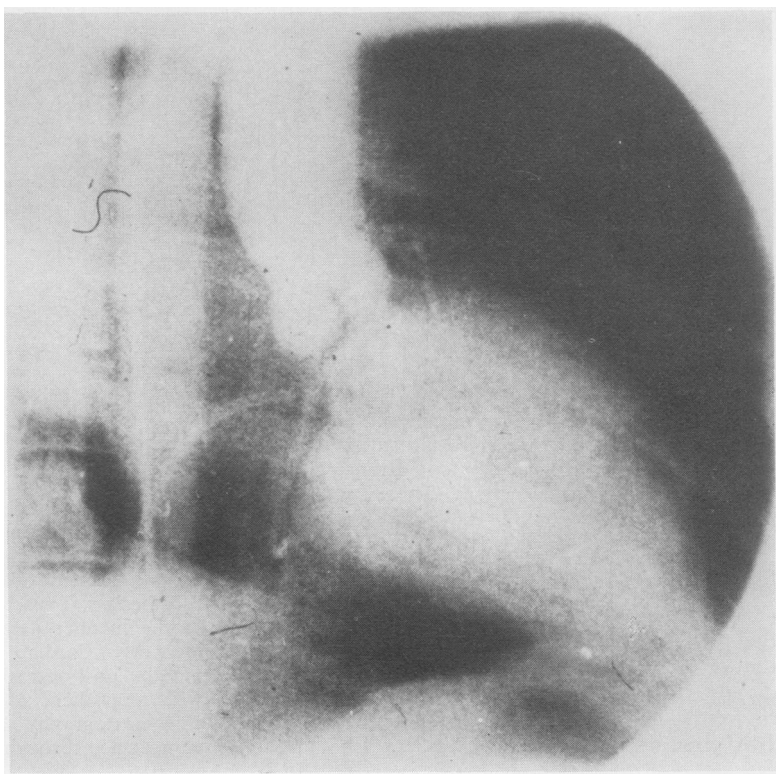


FIG. 7. *Case 4. Ciné angiogram showing some narrowing at the valve ring and a rigid non-calcified valve.*

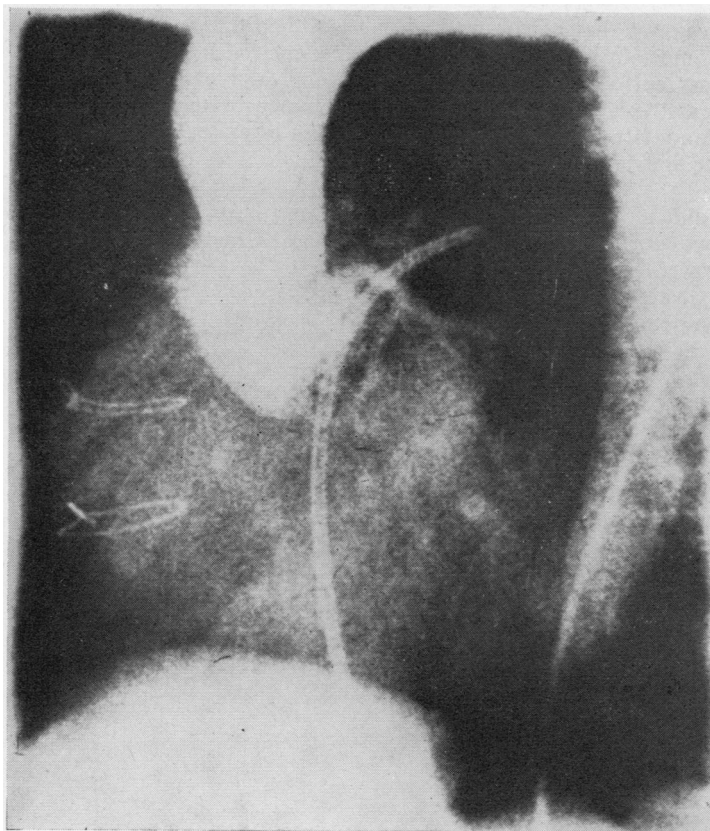


FIG. 8. Case 5. Ciné angiogram showing competent valve in a dilated pocket accommodating the cage.

CASE 4 This patient had calcific aortic stenosis with hypoplasia of the aortic root in association with mitral stenosis. The pre-operative angiogram (Fig. 7) was accepted as showing a small normal aortic annulus with a rigid non-calcified valve. There was a hint of subvalvar muscular hypertrophy.

Under cardiopulmonary bypass mitral valvotomy was performed, and the aortic valve was replaced by a gusset and a size 9A Starr-Edwards prosthesis after aortic inlay insertion. She made a good recovery and remains well and has a competent valve by clinical criteria.

CASE 5 An aortic valvotomy and débridement of a calcified aortic valve had been performed in 1966.

The symptoms of re-stenosis recurred. At the second operation in 1969, the aortic root was enlarged by a gusset and a size 9A Starr-Edwards prosthesis was inserted. She is active and well. Cardiac catheterization post-operatively demonstrated a 17 mm. gradient at rest across the valve.

The ciné angiogram showed the valve to be competent. The dilated pocket accommodating the cage is well demonstrated (Fig. 8).

DISCUSSION

From a study of series of coroners' necropsies which he conducted Laurie (1968) drew attention to aortic root hypoplasia without aortic valve stenosis and accompanied by marked hypertrophy and replacement fibrosis of the ventricular myocardium as a cause for sudden death.

While these cases are not strictly comparable to our five patients with relative hypoplasia of the aortic root accompanied by calcific aortic stenosis, both share a common severe obstructive effect on the ventricular outflow which has led to hypertrophy, and to a risk of sudden death.

In our experience, we have had a higher immediate and late mortality among patients in whom the valve was replaced by the small size 8A Starr-Edwards prosthesis. This contrasts with a good immediate and late recovery in patients receiving the sizes 9 and larger valve (which we recommend).

While it is often difficult to pin-point the cause of death in particular patients, it seems significant that 45% of patients receiving the size 8A valve died either in hospital after operation or over the follow-up period compared with 9% who received the size 9 valve.

Other things being equal, we have found that patients with a large aortic ring, in whom it is technically easier to insert a prosthesis, have a commensurately better immediate and late prognosis after replacement.

The sketches were made by Dr. Jocelyn Farnsworth, of the Fairfax Institute of Pathology, Royal Prince Alfred Hospital, Sydney.

REFERENCE

- Laurie, W. (1968). Aortic hypoplasia as a possible cause of sudden death. *Med. J. Aust.*, 2, 710.